

Leukaemia Section

Short Communication

der(8;17)(q10;q10)

Jean-Loup Huret

jean-loup.huret@atlasgeneticsoncology.org

Published in Atlas Database: August 2018

Online updated version : <http://AtlasGeneticsOncology.org/Anomalies/der0817q10q10ID1801.html>

Printable original version : <http://documents.irevues.inist.fr/bitstream/handle/2042/69761/08-2018-der0817q10q10ID1801.pdf>

DOI: 10.4267/2042/69761

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Abstract

Review on der(8;17)(q10;q10), with data on clinics.

Keywords

Chromosome 8; chromosome 17; TP53 deletion; Chronic lymphocytic leukemia; B-prolymphocytic leukemia; Waldenstrom macroglobulinemia; Acute myeloblastic leukemia.

Disease

Lymphoid and myeloid malignancies. All lymphoid cases were B Lineage. There were 5 cases of chronic lymphocytic leukemia (CLL) (Döhner et al., 1995; Geisler et al., 1997; Adeyinka et al., 2007; Tang et al., 2013), 1 case of B-prolymphocytic leukemia (Schlette et al., 2001), and 1 case of Waldenstrom macroglobulinemia (Wong 2003). There were also 2 myeloid cases; one acute myeloblastic leukemia without maturation (M1-AML) and one acute myeloblastic leukemia with maturation (M2-AML) (respectively Kim et al., 2001 and Mrózek et al., 2002).

Note

der(8;17)(q10;q10) is also a recurrent abnormality in adenocarcinoma of the large intestine.

Epidemiology

The 5 chronic lymphocytic leukemia cases were male patients, aged 50, 84, 92, ?, ?. The B-prolymphocytic leukemia case was a male patient, the Waldenstrom case a female patient aged 80 years, the AMLs cases a male and a female patient aged 68 years.

Prognosis

In only one chronic lymphocytic leukemia case was the survival noted: 52 months of treatment-free survival (Tang et al., 2013).

Cytogenetics

Note

A der(8;17)(q10;q10) is an unbalanced translocation, with 8p and 17 deletions.

Cytogenetics morphological

The der(8;17)(q10;q10) was the sole abnormality in 2 CLL cases, and the sole abnormality within a sideline in the Waldenstrom case (an i(17q) was found in another subclone). A t(14;18)(q32;q21) was present in 2 CLL cases; +12, del(13q), and a complex karyotype were found in 1 CLL case each. The B-prolymphocytic leukemia also had del(7q), del(11q). The 2 AMLs had a del(5q), and a complex karyotype, the M1-AML also presented with del(3p), and the M2-AML with t(10;16)(q22;p13) KAT6B/CREBBP, +19.

Result of the chromosomal anomaly

Fusion protein

Oncogenesis

Genes implicated in oncogenesis in these cases are unknown. Nevertheless it should be noted that the chromosome abnormality results in TP53 deletion.

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This article should be referenced as such:

Huret JL. der(8;17)(q10;q10). *Atlas Genet Cytogenet Oncol Haematol.* 2019; 23(1):14-15.
